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ABSTRACT

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THE USE OF EVENT-RELATED BRAIN POTENTIALS TO MEASURE COGNITIVE PROCESSING DEFICITS IN THE BRAIN-INJURED

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ABSTRACT

Until recently it has been possible only to measure the behavioral products of cognitive processing, eg. reaction time. However, this is a rather indirect way of studying brain substrates of cognition. Psychophysiological techniques can be used to study the neural mechanisms of cognition. In this experiment brain electrical activity was measured and a subcomponent of this electrical activity, the P300 wave, was identified in a population of normal and brain-injured adults while they were engaged in a visual discrimination task. The averaged amplitude of the P300 wave was significantly decreased ($P<0.05$) and the averaged latency of the P300 wave was significantly increased ($P<0.05$) in the brain-injured group. However, a trial-by-trial analysis revealed that near normal waveforms could be produced by the brain-injured subjects in a small percentage of trials. Since the P300 waveform is related to mechanisms of selective attention and short term memory, the appearance of normal P300 waves in the brain-injured group indicates that the stability of these cognitive processes, and not the processes themselves, were affected by the neurological insult to these patients.

Cognitive science is based on the assumption that any incoming information to an organism is transformed, elaborated, organized, stored and retrieved and each of these processes can be inferred and measured by well-controlled experiments. Until recently, however, it has been possible only to measure the products of cognitive processes. Cognition, itself, is inferred from behavioral responses which follow the performance of some task requiring cognitive activity. For example, behavioral measures like reaction time to solve a problem are used in typical experiments to infer certain cognitive processes that take place during the solution to the problem. Clearly, this is a rather indirect way of studying cognition per se. Indeed, the neurophysiological mechanisms underlying complex forms of information processing have yet to be clarified using only behavioral methodologies.

In recent years, cognitive scientists have used psychophysiological techniques to supplement their data collection protocols. This methodology can give unique insights into the neural mechanisms of cognition as they occur. Psychophysiology is concerned with the measurement of physiological responses and how these responses are related to ongoing behavior. One type of physiological response is the measurement of brain electrical activity which is made through the use of scalp sensors during problem solving by a subject. In the last decade there has been tremendous growth in the number of experiments utilizing psychophysiological measures of brain activity to study cognition (for reviews see Beck, 1975; Hillyard & Kutas, 1983;

and John & Schwartz, 1978). Since the brain is the source of cognitive activity, it seems reasonable to assume that the measurement of brain electrical activity, which is certainly an index of brain function, should relate to cognition in some way (Davidson & Erlichman, 1980). Furthermore, by analyzing different patterns of brain electrical activity, cognitive processes which cannot be inferred by observable behavior may be detected (Duffy, 1981; Elliot et al, 1978; Kay et al, 1981). Brain electrical activity is a complex mixture of waves of different frequencies and amplitudes related to both stimuli within the environment and the processing of those stimuli. Thus,during the performance of some task, much of the electrical activity that occurs is not specifically associated with the task, but is random activity or activity that is associated with events to which we are not privy.

Waveform averaging is a method that is used to isolate frequency components which are uniquely correlated to the processing of stimuli (e.g. neuronal activity associated with discrimination between two colors) (Donchin, 1979). As stimuli are repeatedly presented, brain electrical activity is collected and is averaged to previously collected waveforms. Any random brain electrical activity not specifically time-locked to the stimulus event is averaged out and the remainder are the electrical components associated with the stimulus itself and the cognitive processing of that stimulus. These subcomponents of the EEG are referred to as event-related potentials (ERP) and reflect the synchronous activation of aggregates of neurons which correlates to specific aspects of information processing. John (1963) classified ERP's into two types. The first type is considered exogenous in nature. Exogenous evoked potentials are short-latency waveforms which are

evoked by the stimulus and vary with the physical properties of the stimulus. These potentials are sensitive to the functional integrity of specific sensory pathways. The measurements of these exogenous components are used diagnostically in clinical medicine because they are stable and independent of the individual's psychological state. The second type of ERP's are endogenous in nature, are characterized by longer latencies (greater than 100 msec), and are sensitive to the psychological and processing states of the individual. The endogenous event-related brain potentials comprise a family of components, each of which can be correlated to specific aspects of information processing. More specifically, each ERP is a manifestation of brain activity, which plays a role in different facets of information processing (Donchin, 1979). The ERP which has been studied the most extensively is the P300 waveform. The P300 is a positive-going wave which occurs about 300 msec following a stimulus which has some relevance to a subject and thus is evaluated by him/her. The waveform is of greatest amplitude in central parietal regions of the scalp (Simson et al, 1976). It can be elicited using an "oddball" paradigm where frequent and infrequent stimuli are presented to a subject who must discriminate one from the other. The latency of P300 marks the length of stimulus evaluation and the amplitude denotes the degree to which a stimulus was evaluated (Duncan-Johnson & Donchin, 1980). The updating of memory can be considered a part of the stimulus evaluation process of comparing stimuli to be discriminated.

Electrophysiological measurement techniques can be powerful tools because they are sensitive to aspects of information processing which are not normally observable and which may go undetected if traditional behavioral or paper/pencil methods of assessment are used. For example,

if a subject looks at a stimulus for a long while, is it because he wishes to take in more information about the stimulus (the stimulus is interesting), or is he trying to understand it (the stimulus is complex)? An examination of P300 amplitude could give some insight into this question. It has been shown that if P300 amplitude is small and the P300 latency is long, the subject is not evaluating the stimulus actively and the stimulus may be too complex (Donchin et al, 1982). Thus, the P300 represents a neurophysiological index of one aspect of cognitive processing and consequently may be a leading candidate for educational research in cognition.

Disturbances in information processing characterize many cognitively impaired populations. Since psychophysiological measures of endogenous ERP's, like the P300, are sensitive to aspects of information processing as they occur within the brain, these techniques may prove to be useful, supplemental diagnostic tools with populations that show cognitive dysfunctions. For example, Duffy and colleagues have detected differences in P300 scalp topography in groups of dyslexics (Duffy et al, 1980). This finding may be indicative of pathological brain substrates in dyslexics. In schizophrenia, there is a deficiency of processing which is associated with the inability to ignore irrelevant stimuli; that is, to selectively attend to particular stimuli (Chapman & Chapman, 1973). Temporal lobe structures may be related to this function (Ojemann, 1966). A study by Morstyn et al (1983) showed that in schizophrenics the P300 was significantly decreased in amplitude in the left-middle and posterior temporal regions. Thus, there is correspondence between proposed brain areas of dysfunction and the detected electrophysiological anomalies.

Little is known about the relationship of endogenous ERP's to

cognitive processes in patients with closed head-injury. Both Curry (1981) and Campbell (1982) demonstrated reduced P300 amplitude and increased P300 latency in a closed head injury group up to 22 months post injury although their patients responded satisfactorily on the auditory discrimination task they were asked to perform. In view of these findings, the lack of a P300 wave may be useful as a diagnostic sign of severe head injury. Behaviorally, there seems to be an inability to ignore relevant stimuli in these patients. Research indicates that the P300 may be exquisitely sensitive to this cognitive dimension.

This study attempts to utilize the technique of recording brain event-related potentials of brain-injured subjects engaged in a cognitive task. The task will be more difficult than tasks previously used with this population. In addition, the electrical information from the brain will be critically analyzed in a way which gives new insights about the learning process of brain-damaged adults. Suggestions regarding a proposed remediation technique will also be discussed.

METHOD

Subjects

A total of 20 healthy male undergraduates (mean age = 24.0 years; range 18-42 years) with no history of head injury and recruited from the Industrial Technology Education program at The Ohio State University were used as subjects in this experiment. Eye blink and other muscle artifacts exceeded an á priori criterion of acceptance in eight subjects. Thus, data were analyzed from only 12 subjects (mean age = 23.4 years). Six subjects were classified as left-handed and the remaining six as right-handed on the basis of the results of the Edinburgh Handedness Inventory (Oldfield, 1970). In addition, in order to demonstrate the

utility of this technique to detect information processing deficits in brain-injured populations, a total of six brain-injured subjects (three male and three female; mean age 33 years; range 16-42 years) were tested. These subjects were either patients of The Ohio State University Hospitals (three males) or outpatients of a private practice psychologist in the Columbus, Ohio area. Each patient had a history of either closed head injury due to rapid deceleration in an auto accident (n=4), stroke (n=1), or aneurysm (n=1), all of which resulted in temporal lobe pathology. In addition, each patient had excellent distal motor control with no sign of resting or intention tremor and all were right handed.

Apparatus

An Apple II+ microcomputer, with 48K memory and two disk drives were used in conjunction with an Isaac 91A interface (Cyborg Corp.) to deliver stimuli to subjects via a color monitor (Amdek). Stroop stimuli (Stroop, 1935) were represented on the Apple low resolution graphics screen. One hundred stimulus items were given in each of four blocks to each subject. Within each block there were equivalent numbers of the words: red, blue, and yellow which always were displayed in an interfering "ink" color (either red, blue or yellow). The blue "ink" color was designated the "oddball" target stimulus and was presented 16% of the time. Each time a target stimulus appeared, a signal marker was delivered to an FM recording adaptor (Vetter), which was then put onto cassette tape along with the on-going electrical activity from the brain. Brain electrical response data were amplified using a Beckman Accutrace eight channel polygraph. The computer software control program generated and delivered the stimuli by activating the low resolution graphics screen for 64 msec. When the screen was turned on

the subject was exposed to the stimulus and when a target stimulus occurred, a timer started. The subject responded to each target by pressing a switch on a response panel held on his lap which stopped the timer and stored the response latency.

Procedure

Initially, each subject was asked to sign a form giving consent to record brain activity with scalp sensors. The subject was seated in a quiet room directly in front of a video monitor (36 inches from screen to eyes). A response panel containing two buttons was placed on the subject's lap and an electrode cap was fitted to the head with conductive gel placed at electrode sites Fz (ground) and Pz (active lead) using the 10-20 International System of Electrode Placement (Jasper, 1958). In addition, the left earlobe was fitted with an electrode that served as a reference. Instructions were displayed to the subject via the video monitor and were read to him by the experimenter. The subject was instructed to look at the screen during the session, not to speak or make any sudden movements of the head and/or body. Furthermore, the subject was told to ignore all colors except blue. When he saw the color of blue, the subject was told to press a response button with the index finger. A practice series of six trials were then given with hand-held Stroop cards in which the subject responded to the blue ink color. He was then given the opportunity to press the response buttons as many times as were required to eliminate arm or torso movement associated with the press. Any questions regarding the protocol were answered at this time. Subjects were told that they would be monitored by a video camera. A pair of headphones equipped with white noise were placed over the subject's ears (to eliminate extraneous

noise). Overhead lights were turned off and two 300 volt bulbs (located on the floor) were used to illuminate the room enough to eliminate screen reflections and the door was closed. One minute later the first block of 100 stimulus trials was presented, each containing 16 target (blue color) trials presented randomly by the Apple computer. The same random sequence of stimulus presentation was used in each of four total blocks of trials and for each subject. The time between each trial was two seconds. Two minutes of rest were given between each block of trials. In addition to brain electrical activity from Pz, reaction time (button pressing) to Stroop target stimuli was also recorded. Subjects responded in an ABBA hand sequence across each block of trials and the starting hand was balanced across subjects within each group. Finally, subjects were de-briefed after the experimental session and any questions about the experimental hypotheses were answered.

Analysis of Waveforms

The EEG analysis procedure consisted of playing back the previously recorded cassette tape of brain electrical response data into an A/D channel of the Isaac interface. The brain activity that occurred after each signal marker, which designated target stimulus onset, was summed and averaged to any previous target waveform. A total of 25 waveforms for each subject were averaged in this manner using the Apple-Isaac computer system and a graph was generated for each subject with an Epson (MX80-FT) printer.

Rejection of artifact was accomplished by setting low and high pass filters at 0.5 Hz and 40 Hz respectively. Signals greater than $\pm$40 microvolts in amplitude were filtered out in order to eliminate eye blink artifact that might occur simultaneously with a target stimulus.

Waveform measurements of amplitude and latency of the P300 component were obtained for each subject. The P300 wave was identified as the most positive-going peak between 250 and 500 msec after target stimulus onset.

RESULTS

The P300 graphs for each brain-injured subject and two normal subjects are shown in Figure 1. The P300 amplitude and P300 latency and reaction time data from the brain-injured subjects were compared with the normal subjects. Figure 2 shows P300 latency, P300 amplitude, and reaction times, respectively, for all subjects in both the normal and brain-injured groups. The Mann-Whitney-U test was used to test for significance between the groups on these measures. There was a signif'-cant difference in P300 latency ($Z=3.14$; $P<0.05$), P300 amplitude ($Z=2.53$; $P<0.05$), and reaction time ($Z=2.90$; $P<0.05$) between the two groups. Thus, the results support the data of both Curry (1981) and Campbell (1982) that P300 amplitude is reduced and P300 latency is increased in individuals with closed head injury. Furthermore, this study demonstrates that the deficits exist for a visual discrimination task as well.

The data were analyzed for both the normal and the brain-injured groups on a trial-by-trial basis. This was done to detect the trial-by-trial occurrence of the P300 ERP. For artifact-free trials only, a determination was made as to whether the P300 existed in each epoch. In a sample of normal and brain-injured subjects it was found that the P300 occurred 96% of the time in normals and only 22% of the time in the brain-injured group.

DISCUSSION

The brain-injured group performed the Stroop task as accurately as the controls (95% correct), but the data indicate that they may have

had greater difficulty with the discrimination task than the controls. Not only were their reaction times elevated, which supports this conclusion, but the P300 waveforms, when they did exist, had a much longer latency and lower amplitude. The longer P300 latency indicates longer evaluation time was required for the discrimination. Also, the reduced P300 amplitude indicates that little confidence was shown to the task solution or little memory updating was occurring during the discrimination task.

One possible source of the generator potential of the P300 wave is the hippocampus and temporal lobes (Halgren et al, 1980), which are associated with short-term memorial processes (Ojemann, 1966). This area of the brain would be essential to the comparing of two stimuli and the updating of memory with the most recent results of the comparison. The data indicate that the P300 may be a sensitive index of dysfunction to this system in the brain injured. Indeed, some of these subjects were involved in accidents of rapid deceleration (head strikes dashboard of car during sudden stop) which can damage temporal lobe structures. During rapid deceleration of an auto, the forehead typically strikes a solid object causing the inferior temporal lobes to move rapidly across the sphenoid bones resulting in tissue damage. Thus, difficulty may result when using this short term memory mechanism to do the task of comparing two colors. The data support this conclusion.

On the other hand, five of six of the individuals in this group were taking varying doses of Dilantin (n=4; 300-400 mg/day) or Nembutal (n=1; 100 mg/day) prophylactically for the control of seizures and Nembutal is known to decrease arousal levels which could affect P300 amplitude. Interestingly, the subject who was not on any medication

showed the smallest amplitude of P300 of all subjects while the subject on Nembutal showed the second highest P300 amplitude of the brain-injured subjects. However, further studies will be needed to determine more conclusively if these results were contaminated by drug effects. Whatever the case, it can be reasonably stated that the amount of active stimulus evaluation that occurred during the visual discrimination task was less in the brain-injured group and this psychophysiological technique was sensitive enough to detect the phenomenon.

At first glance the conclusion that the brain-injured group evaluates stimuli to a lesser extent than normals seems reasonable. However, the method of trial-by-trial analysis used in this study has shed some new light on this interpretation. Using this technique, each artifact-free target epoch was examined individually. The number of trials that show evidence of P300 waveform through visual examination by the experimenter was noted. It was shown that in some brain-injured subjects, in a small percentage of trials, robust P300 waves existed, while in normal subjects evidence of a P300 appeared on almost every trial. The fact that normal-looking P300 waves appear at all in the brain-injured group is surprising since the average waveforms for some individuals were virtually flat (see fig. 1). The appearance of P300 waves indicates that the brain-injured subjects were not as consistently engaged in the task as were normals. This could be due to the moment-by-moment waxing and waning of attention because of either distractions or other factors related to poor mechanisms of selective attention. The stability of the process and not the process itself seems to be affected. Whatever the reason, this group cannot consistently maintain active involvement with most stimuli encountered during the session. Thus, this trial-by-trial

analysis technique offers a new interpretation of this study and other similar studies that measure P300 in populations of brain injured. Both P300 latency shifts from trial-to-trial or the absence of P300 waveforms in any given trial can be a source of between-subject or between-group differences in the average ERP waveform. In fact, if one is averaging only 20-25 trials, which is typical, one or two trials without a P300 wave in a given subject can account for a 5-10% difference in amplitude between that subject and another who always shows P300. Can we then correctly conclude that an attenuated, average P300 wave is due to a consistently attenuated processing difference? More reasonably, the difference may result because of a momentary lapse of attention for that subject.

CONCLUSION

Considering the results of this and related studies (Campbell, 1982; Curry, 1981; Duffy et al, 1980; Morstyn et al, 1983), it may be possible in the near future to use measures of brain activity as both a diagnostic aid and a rehabilitation tool to supplement current methods of assessment and treatment of both cognitively-impaired and normal learners. Additionally, it should be possible to monitor any changes in brain electrical activity that occur in an individual as a result of cognitive remediation therapy. Thus, this technique could provide the clinician with a measurable record of progress. It is hoped that measures of brain electrical activity can provide criteria for strategies of cognitive rehabilitation. For example, given these results, one rehabilitation or teaching strategy to consider for brain-injured clients involves the use of biofeedback. Joel Lubar at the University of Tennessee (Lubar & Deering, 1981) has reported impressive gains in training children with

learning disabilities, who show slower prefrontal brainwave activity, to increase the frequencies of brainwaves in these areas. Furthermore, he has demonstrated that once these children can successfully increase beta activity in frontal cortical areas, this physiological change can be correlated with improved performance on problem-solving tasks which were once difficult for the child.

It is suggested that providing auditory biofeedback to a client, (with reduced P300 incidence) when a P300 has been produced during a problem-solving task, will help the client develop a cognitive strategy that may lead to an ability to increase the frequency of P300 waveform production during a task. This should then correlate to a cognitive set that may improve attention and facilitate active processing of stimuli, which could consequently improve consolidation of information into long-term memory stores.

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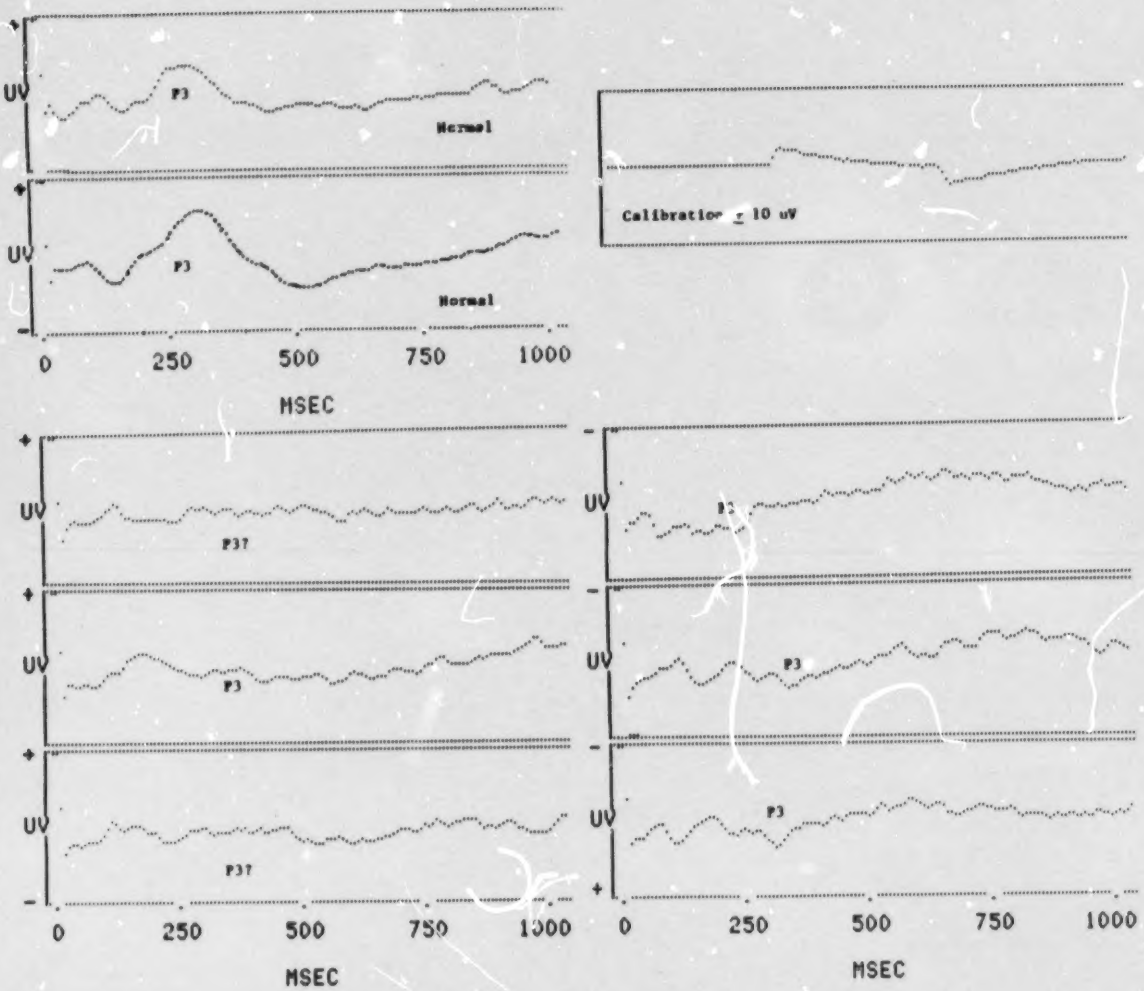
LEGENDS

Figure 1. Graphs of 25 averaged trials per subject showing two normal subjects and their P300 (P3) waves, six brain-injured subjects and their attenuated P300 (P3) waves and a calibration curve for amplitude reference.

Figure 2. Top graph is P300 latency for all subjects. Middle graph is P300 amplitude for all subjects. Bottom graph is reaction times for all subjects.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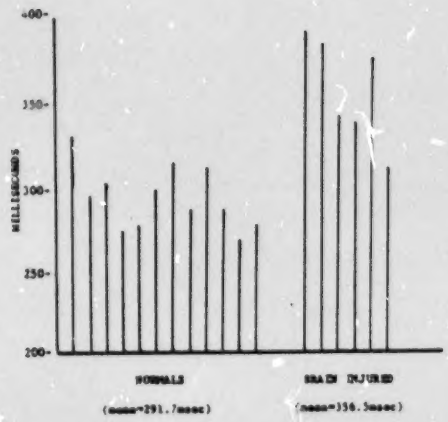
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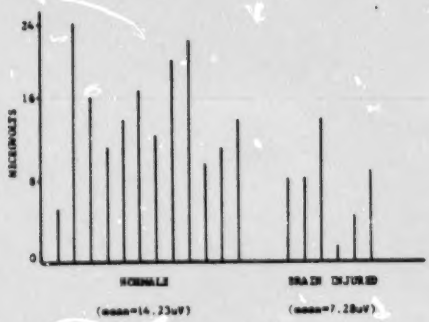
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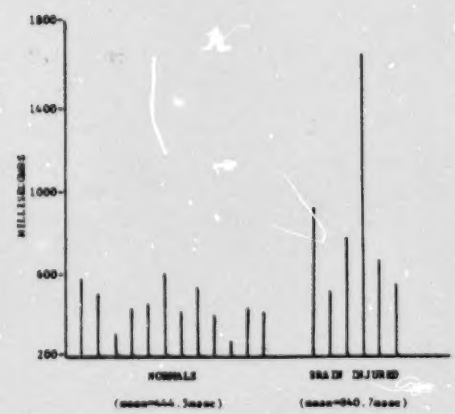
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NORMALS
BRAIN INJURED
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